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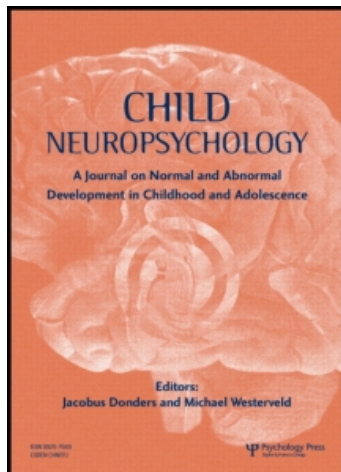
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# Response Inhibition in Children With DSM-IV Subtypes of AD/HD and Related Disruptive Disorders: The Role of Reward

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## ABSTRACT

The current study had four aims: (a) to replicate previous findings of slow response inhibition in Attention Deficit/Hyperactivity Disorder (AD/HD), (b) to explore whether poor response inhibition in children with AD/HD is a core problem or rather a result of an underlying problem related to reward, (c) to investigate the specificity of poor response inhibition and the role of reward in relation to AD/HD, and (d) to study whether findings would be different for three subtypes of AD/HD. In order to address these issues, a stop paradigm was administered under a reward condition and under a nonreward condition to an AD/HD group ( $n = 24$ ), an Oppositional Defiant Disorder (ODD)/Conduct Disorder (CD) group ( $n = 21$ ), a comorbid AD/HD + ODD/CD group ( $n = 27$ ), and a normal control (NC) group ( $n = 41$ ). Firstly, contrary to prediction, none of the Disruptive Behavior Disorder (DBD) groups differed from the NC group with respect to the speed of the inhibition process. Secondly, it was shown that children with AD/HD and children with comorbid AD/HD + ODD/CD, but not children with ODD/CD alone, slowed down more dramatically in the reward condition than normal controls. This finding was interpreted as a strategy to increase the chance of being rewarded in children with AD/HD and children with comorbid AD/HD + ODD/CD, but not in children with pure ODD/CD. Finally, analysis of AD/HD subtypes did not change the main findings of this study.

An influential point of view on the nature of AD/HD is that children with AD/HD primarily suffer from suboptimal energetic states (Sergeant, Oosterlaan, & Van der Meere, 1999; Sergeant & Van der Meere, 1990a; Van der Meere, 1996). Research applying the cognitive energetic model (Sanders, 1983, 1998) to task performance in children with AD/HD suggests that children with AD/HD have specific problems with the output stages of information processing and with the energetic pools activation and/or effort (Douglas, 1999; Leth-Steensen, Elbaz, & Douglas, 2000; Sergeant et al., 1999). An optimal activation state

is a prerequisite to prepare for motor action. The effort pool has the task of maintaining an optimal state of arousal and activation to meet the demands of the task to be performed. Motivational variables such as feedback and reward are strongly related to effort allocation (Sanders, 1983, 1998). Several researchers have hypothesized that a core problem of children with AD/HD is an unusual sensitivity to reward (Douglas, 1999; Haenlein & Caul, 1987; Wender, 1972). In this paper we will use the term 'reward deficit' to refer to general deficits of reward, motivation, or effort, due to the highly related nature of these variables.

The neural circuitry suggested to be underlying the processing of reward information involves the dopamine neurons of the ventral tegmental area (VTA) and substantia nigra, which are connected to brain structures involved in motivation such as the striatum, nucleus accumbens, and frontal cortex (Schultz, Dayan, & Montague, 1997). Sagvolden and Sergeant (1998) suggested that the meso-limbic dopamine branch plays a significant role in reinforcement in AD/HD. Recently, an event-related fMRI study on reward provided evidence for the involvement of the basal ganglia (particularly the striatum) in the processing of reward-related information (Delgado, Nystrom, Fissell, Noll, & Fiez, 2000). Elliott, Friston, and Dolan (2000) found that the level of reward was related to activity in the midbrain and ventral striatum (which is a crucial component of dopaminergic projection systems). Koepp et al. (1998) reported increased dopamine release in the striatum during performance of a financially rewarded video game.

Support for the reward hypothesis comes from studies showing that children with AD/HD are prone to giving up on effortful tasks (e.g., Borcharding et al., 1988; Milich & Okazaki, 1991). Several researchers have suggested that performance of children with AD/HD is dependent on the presence or absence of response contingencies (e.g., Douglas, 1985; Haenlein & Caul, 1987). The importance of reward mechanisms in AD/HD also stems from theoretical explanations of the effects of stimulant medication in children with AD/HD (Wilkison, Kircher, McMahon, & Sloane, 1995). Wilkison et al. interpreted their findings in terms of methylphenidate increasing the reward value of reinforcers in children with AD/HD. Although researchers seem to agree that an unusual sensitivity to reward is a characteristic of children with AD/HD, disagreement exists as to whether these children are over-sensitive or under-sensitive to reward. Some researchers argue and have shown that children with AD/HD are less sensitive to reward (Haenlein & Caul, 1987; Wender, 1971, 1972). Others have argued and shown that children with AD/HD evidence an increased tendency to look for immediate reward (e.g., Douglas & Parry, 1994; Tripp & Alsop, 1999). Note that in Tripp and Alsop's study reward was

not given for all correct responses. For the correct responses that were rewarded, reward was given immediately. In Douglas and Parry's study, reward was not contingent on the child's performance, but administered randomly. Reward was given immediately. There are also studies that failed to demonstrate that reward differentially affects the performance of children with AD/HD, when compared with normal children (Iaboni, Douglas, & Baker, 1995; Oosterlaan & Sergeant, 1998a). Both these studies provided the children with reward immediately after a correct trial. Although there is disagreement as to whether children with AD/HD are less or more sensitive to reward, the results of several studies have suggested that children with AD/HD react differently to reward compared with control children.

Another influential theoretical account for AD/HD stems from Barkley (1997). He proposed that a deficit in behavioral response inhibition (as a primary executive function) is the core dysfunction in AD/HD and is specifically related to this disorder. In order to measure the ability to inhibit a response in AD/HD, the stop paradigm (Logan & Cowan, 1984) has been used in several studies (Oosterlaan, Logan, & Sergeant, 1998; Tannock, 1998). The stop paradigm enables measurement of the latency of response inhibition ("stop") and response execution ("go") independently of one another. In most stop paradigm studies, children with AD/HD showed slow response inhibition (Nigg, 1999; Oosterlaan & Sergeant, 1998a, 1998b; Pliszka, Borcharding, Spratley, Leon, & Irick, 1997; Rubia, Oosterlaan, Sergeant, Brandeis, & van Leeuwen, 1998; Schachar, Mota, Logan, Tannock, & Klim, 2000; Schachar, Tannock, Marriott, & Logan, 1995; see for reviews Oosterlaan et al., 1998; Sergeant et al., 1999).

Recently, it was suggested that the prefrontal cortex and possibly the globus pallidus are involved in response inhibition (Band & Van Boxtel, 1999). Casey et al. (1997) reported significant correlations between inhibitory performance and volumetric MRI measures of the prefrontal striatal circuit (prefrontal cortex, nucleus caudate, and globus pallidus). Importantly, only the correlation with the prefrontal cortex volume was unique to inhibitory processes. Rubia et al. (2001) showed that during performance of the stop paradigm,

activation was observed in the right anterior cingulate, supplementary motor area, inferior prefrontal, and parietal cortices.

The majority of studies on response inhibition in AD/HD have employed a stop paradigm with fixed intervals between the go and the stop stimulus (Oosterlaan et al., 1998). However, the use of a tracking mechanism in order to vary the delay between go and stop stimulus has several theoretical and practical advantages over the stop paradigm with fixed intervals (Band, 1997). In the current study, the stop paradigm with a tracking mechanism was used. To date, only four studies on inhibition in AD/HD have employed the tracking mechanism version of the stop paradigm (Chhabildas, Pennington, & Willcutt, 2001; Nigg, 1999; Schachar et al., 2000; Scheres, Oosterlaan, & Sergeant, 2001).

In addition to AD/HD, ODD and CD have been associated with a deviant sensitivity to reward and with a deficit in response inhibition. For example, Shapiro, Quay, Hogan, and Schwartz (1988) showed that children with CD were more sensitive to reward than normal control children. Quay (1988) argued for an overactive reward system in CD. However, studies testing the reward hypothesis in CD have produced conflicting results (see for review Quay, 1993). There are only two studies that addressed the specificity issue of reward dominance in children with AD/HD and ODD or CD (O'Brien & Frick, 1996; Oosterlaan & Sergeant, 1998a). Poor inhibitory performance has been suggested to be related to CD. Quay (1993) predicted that children with antisocial behavior would show poor response inhibition resulting from a relatively overactive reward system in combination with a relatively underactive inhibition system. In some studies it has been shown that slow response inhibition is specifically related to AD/HD (Schachar & Logan, 1990; Schachar & Tannock, 1995; Schachar, Tannock, & Logan, 1993). However, a meta-analysis demonstrated deficient inhibitory control in both children with AD/HD and children with ODD/CD (Oosterlaan et al., 1998).

The purpose of the current study was to bridge two important theoretical accounts of AD/HD, that is, poor response inhibition as the core deficit, and an unusual sensitivity to reward as

the main deficit of the disorder. It is unclear whether poor response inhibition in AD/HD is the core symptom, or a manifestation of an underlying reward deficit. Therefore, the current study aimed at (a) replicating poor response inhibition in AD/HD using a stop paradigm with tracking mechanism (as opposed to fixed intervals), (b) exploring whether poor response inhibition is a core problem in children with AD/HD, or, alternatively, whether it is a manifestation of an underlying reward deficit, and (c) examining the *specificity* of deficits in response inhibition and the specificity of the role of reward in response inhibition by comparing three groups of DBD children (pure AD/HD without comorbid ODD/CD, pure ODD/CD without comorbid AD/HD, and comorbid AD/HD + ODD/CD) with normal children. Finally, since there is a debate on which subgroups should be placed under a DSM-IV AD/HD diagnostic category (Barkley, 1997; Milich, Balentine, & Lynam, 2001), it was studied here whether children of different AD/HD subtypes performed differently.

In order to address the four aims of the current study, a stop paradigm with a tracking mechanism (Logan, 1994; Osman, Kornblum, & Meyer, 1986) was administered under a reward condition and under a nonreward condition. In the reward condition, reward was given immediately after a successful inhibition trial. In the nonreward condition, subjects received no reward. The subjective motivation level of the children was measured in both conditions. If poor response inhibition is a core problem in AD/HD, then an inhibition deficit will be observed in both the nonreward and reward conditions. If a reward deficit is responsible for the deficit in response inhibition in AD/HD, the response inhibition deficit will be most pronounced in the nonreward condition compared with the reward condition (a group by condition interaction is expected).

## METHOD

### Subjects and Selection Criteria

One hundred and fifteen children in the age range of 7–12 years participated in this study. The participants were assigned to one of four groups, that is, the normal

control group (NC), the AD/HD group, the ODD/CD group, or the comorbid (AD/HD and ODD/CD) group. The three psychopathological groups were selected from 14 special educational services, which are specialized in the education of children with extreme behavioral problems. Of all Dutch children in the age range of 6–12 years, 2.2% attend these special educational services (Central Office for Statistics, personal communication). The normal control children were selected from six regular schools. Schools were located throughout the country.

In order to select participants, a two-stage procedure was used. In the first stage, 1504 households (876 parents of children who were placed in special schools and 628 parents of children in regular schools) received information on the study, an informed consent form, and 2 child behavior questionnaires. If parents were willing to participate, they signed the informed consent form and completed the questionnaires. Questionnaires were the Disruptive Behavior Disorder Rating Scale (DBD; Pelham, Gnagy, Greenslade, & Milich, 1992; Oosterlaan, Scheres, Antrop, Roeyers, & Sergeant, 2000) and the Child Behavior Checklist (CBCL; Achenbach 1991; Verhulst, Van der Ende, & Koot, 1996). The DBD consists of: (a) two subscales composed of the DSM-IV items for AD/HD, that is, an Inattention subscale and an Hyperactivity/Impulsivity subscale, (b) a scale composed of the DSM-IV items for ODD, and (c) a scale composed of the DSM-IV items for CD. Items were rated on a scale ranging from 0 to 3. The DBD was used to select participants for the study. The major advantage of this rating scale is that it includes statements listed as behavioral descriptors of AD/HD, ODD, and CD in the DSM-IV. Parents of 576 children completed the questionnaires (response rate 38.3%). There were 337 children who met the inclusion criteria for one or more of the four groups (see below), and these children entered the second stage.

At stage two, teachers completed the DBD, the Teacher Rating Form (TRF; Achenbach, 1991; Verhulst et al., 1996) and the IOWA Conners Teacher Rating Scale (IOWA CTRS; Oosterlaan, Prins, & Sergeant, 1992; Pelham, Milich, Murphy, & Murphy, 1989). Three hundred and two sets of completed questionnaires were received (response rate 89.6%).

For a child to be included in one of the three psychopathological groups both parent and teacher ratings had to meet inclusion criteria for that particular group. In this way the criterion of pervasiveness of the disorder was met. The inclusion criteria used were based on the DSM-IV symptoms for AD/HD, ODD, and CD. Inclusion criteria for the AD/HD group were: a rating of 12 or more on the Inattention subscale and/or on the Hyperactivity/Impulsivity subscale of both the parent and the teacher DBD.

- AD/HD inattentive subtype was defined as: (a) a rating of 12 or more on the Inattention subscale of both the parent and the teacher DBD, and (b) a rating lower than 12 on the Hyperactivity/Impulsivity subscale by at least 1 informant.
- AD/HD hyperactive/impulsive subtype was defined as: (a) a rating of 12 or more on the Hyperactivity/Impulsivity subscale of both the parent and the teacher DBD, and (b) a rating lower than 12 on the Inattention subscale by at least 1 informant.
- AD/HD combined subtype was defined as: (a) a rating of 12 or more on the Hyperactivity/Impulsivity subscale of both the parent and the teacher DBD, and (b) a rating of 12 or more on the Inattention subscale of both the parent and the teacher DBD.

To be included in the ODD/CD group, the following criteria had to be met: (a) a rating of at least 8 on the ODD scale or a rating of at least 6 on the CD scale of the parent DBD, and (b) a score of at least 8 on the ODD scale or a score of at least 6 on the CD scale of the teacher DBD. To be assigned to the comorbid group, the criteria of both the AD/HD group and the ODD/CD group had to be met. In order to exclude children with psychotic symptoms, an additional criterion for all three psychopathological groups was that the child was rated at or below the 75th percentile on the Thought Problem scale of the CBCL and the TRF.

To be assigned to the NC group both parents and teachers were required to rate the child (a) below the critical values of all the scales of the DBD, (b) at or below the 75th percentile on all the scales of the CBCL and the TRF, and (c) below the suggested cut-off scores on the Inattention/Overactivity scale and the Oppositional/Defiant scale of the IOWA CTRS (Pelham et al., 1989).

There were 154 children who met the criteria for membership of one of the four groups. However, 39 children did not participate in the study for various reasons. The most important reason for exclusion at this stage was use of medication that could not be discontinued (pipamperon or clonidine:  $n = 20$ ). Twenty children were excluded from the study because they used medication that might have interfered with the performance on the stop paradigm. Other children dropped out because of moving house, finishing school, or parents who withdrew their consent. The remaining 115 children participated in the experiment. Five AD/HD children, 5 comorbid children and 1 ODD/CD child used methylphenidate (Ritalin<sup>®</sup>), but discontinued temporarily the use of this medication at a minimum of 18 hr prior to the experiment.

Two children were excluded prior to data analyses: one because of an extreme low IQ ( $IQ = 48$ ), and the other because of a diagnosis of Asperger syndrome. The groups consisted of 24 AD/HD children, 21 ODD/CD children, 27 comorbid children, and 41 normal

control children. The AD/HD group consisted of 9 pervasively inattentive subtype children, 6 pervasively hyperactive/impulsive subtype children, 7 pervasively combined subtype, and 2 children who were defined as inattentive by one rater and hyperactive/impulsive by the other rater.

Eight children who were assigned to the ODD/CD group appeared to be children with CD (a rating of at least 8 on the ODD scale by *both* raters and a rating of at least 6 on the CD scale by a single rater), and 13 children in this group met the criteria for ODD.

In the comorbid group, 12 children met the criteria for AD/HD and CD (a rating of at least 8 on the ODD scale by both raters and a rating of at least 6 on the CD scale by one rater), 14 children met the criteria for AD/HD and ODD (a rating of at least 8 on the ODD scale by both raters), and 1 child met the criteria for AD/HD and CD (without meeting the criteria for ODD). The distribution of subtypes AD/HD in the comorbid group was as follows: 10 pervasively inattentive subtype children, 3 pervasively hyperactive/impulsive subtype children, 12 pervasively combined subtype, and 2 children who were defined as inattentive by one rater and hyperactive/impulsive by the other rater.

A Student Newman Keuls procedure (overall  $\alpha$  set at .05) showed that the groups did not differ with respect to age. The NC group had fewer male subjects and a higher mean IQ than the other groups (see Table 1). Correlations showed, however, that the dependent variables of the stop paradigm were significantly correlated only with age. Each of the three psychopathological groups could be distinguished from one another and from the NC group on the DBD scales that were used as the criterion measures. In addition, the selected groups differed from one another on a number of other scales. As would be predicted, the AD/HD group and the comorbid group showed the highest scores on the Attention scale of the CBCL and the TRF, and on the Inattention/Overactivity scale of the IOWA CTRS. As predicted, the ODD group and the comorbid AD/HD+ODD group, showed the highest scores on the Aggression and Delinquency scales of the CBCL and the TRF, and on the Oppositional/Defiant scale of the IOWA CTRS (see Table 1). This supports the behavioral distinctiveness of the four groups.

### Stop Paradigm

The stop paradigm involves two types of trials: go trials and stop trials. Go trials were airplanes, presented for a period of 300 ms at the midpoint of the computer screen. Immediately before the go stimulus onset, a fixation point (200 ms in duration) appeared on the screen. If the airplane pointed to the right, subjects were required to press the right response button. If the plane pointed to the left, subjects were instructed to press the left button. Stop trials consisted of a go trial and a stop

signal (a 1000 Hz tone, 50 ms in duration), presented through earphones. The stop signal was usually presented shortly after the airplane, but could also be presented concurrently with or shortly before the airplane, dependent on the child's performance (see below). Children were instructed not to press either of the two buttons when the plane was followed by the tone – 75% of the trials were go trials, and 25% were stop trials. The stop paradigm allows measurement of both response execution (go trials) and response inhibition (stop trials).

Trials were presented in blocks of 32 trials. Within a block the plane pointed equally often to the right or to the left. Stop signals were balanced for right and left go trials. Stop trials were presented randomly within each block with the restriction that two stop trials were presented in succession only once in each block.

The task commenced with four practice blocks, to make sure that the children were familiar with the paradigm. In the first two practice blocks only go trials were presented. During practice of the go task, children were encouraged with standardized instructions to respond as quickly as possible without making too many errors. In the last two practice blocks, 25% of the trials were stop trials. During practice of the stop task, children were instructed to work as quickly as possible and to try to inhibit their response when they heard the stop signal. After the practice blocks, participants were administered six experimental blocks of 32 trials. To examine the effect of reward on response inhibition, the task was administered in two conditions: one with reward and one without reward. In each condition, children were presented with three blocks of 32 trials.

### Dependent Variables and the Race Model

The main dependent variable in the stop paradigm was stop signal reaction time (SSRT), which reflects the latency of the inhibitory process. SSRT cannot be observed, because the response to a stop signal is a covert one. Therefore, SSRT has to be estimated. This can be done using the race model (Logan & Cowan, 1984). This model assumes that the go process and the stop process are independent. The go stimulus triggers the go process and the stop signal initiates the stop process. The process that finishes first wins the race. If the go process wins the race, the response is executed. If the stop process finishes first, the response is inhibited. The outcome of the race depends on the speed and the variability of the go process, the delay between go stimulus and stop signal, and the speed and the variability of the stop process. In the present study, a tracking mechanism was used to vary dynamically the delay between go and stop signal, contingent on the subject's performance (Logan, Schachar, & Tannock, 1997; Osman et al., 1986). The initial delay between go

Table 1. Means, Standard Deviations, and Pairwise Group Comparisons for IQ, Age, and Rating Scale Scores.

Measure	Group								Pairwise group comparisons <sup>b</sup>
	AD/HD (a) <i>n</i> = 24(18) <sup>a</sup>		ODD/CD (o) <i>n</i> = 21(19) <sup>a</sup>		Comorbid (c) <i>n</i> = 27(25) <sup>a</sup>		NC (n) <i>n</i> = 41 (24) <sup>a</sup>		
	<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>	
IQ	92.2	15.1	86.9	14.7	82.7	12.6	105.0	25.0	<i>n</i> > <i>a</i> , <i>o</i> , <i>c</i>
Age	10.1	1.5	10.7	1.3	10.9	1.5	10.2	1.6	<i>ns</i>
CBCL									
Attention	70.6	6.6	64.7	8.4	72.3	7.8	51.0	1.9	<i>a</i> , <i>c</i> > <i>o</i> > <i>n</i>
Aggressive <sup>c</sup>	65.3	7.6	70.8	8.6	74.6	9.4	50.2	0.8	<i>o</i> , <i>c</i> > <i>a</i> > <i>n</i>
Delinquent <sup>d</sup>	59.5	7.2	65.1	8.4	67.8	7.8	50.4	1.2	<i>o</i> , <i>c</i> > <i>a</i> > <i>n</i>
TRF									
Attention	62.7 <sup>e</sup>	4.9	56.9	4.5	63.0	4.8	50.3	1.0	<i>a</i> , <i>c</i> > <i>o</i> > <i>n</i>
Aggressive <sup>c</sup>	62.8 <sup>e</sup>	6.8	68.0	7.4	74.7	11.0	50.3	0.9	<i>c</i> > <i>o</i> , <i>a</i> > <i>n</i>
Delinquent <sup>d</sup>	56.6 <sup>e</sup>	6.6	63.2	6.1	67.4	10.7	50.7	1.8	<i>c</i> > <i>o</i> , <i>a</i> > <i>n</i>
DBD parents									
Inattention	14.7	5.1	12.0	5.1	14.9	4.6	1.6	2.3	<i>a</i> , <i>o</i> , <i>c</i> > <i>n</i>
I/H <sup>f</sup>	15.0	5.4	12.0	3.9	15.1	4.5	1.5	1.8	<i>a</i> , <i>c</i> > <i>o</i> > <i>n</i>
ODD	8.1	3.9	13.3	3.8	12.7	3.7	1.5	1.9	<i>o</i> , <i>c</i> > <i>a</i> > <i>n</i>
CD	2.0	2.2	3.6	3.7	3.9	2.7	0.2	0.5	<i>o</i> , <i>c</i> > <i>a</i> > <i>n</i>
DBD teacher									
Inattention	14.8	3.8	7.3	3.1	14.8	3.9	0.6	1.2	<i>a</i> , <i>c</i> > <i>o</i> > <i>n</i>
I/H <sup>f</sup>	12.6	5.3	7.3	4.3	13.9	4.6	0.5	1.4	<i>a</i> , <i>c</i> > <i>o</i> > <i>n</i>
ODD	6.8	5.3	12.6	4.3	14.0	4.4	0.1	0.4	<i>o</i> , <i>c</i> > <i>a</i> > <i>n</i>
CD	2.0	2.8	4.5	3.5	5.5	4.8	0.0	0.0	<i>o</i> , <i>c</i> > <i>a</i> > <i>n</i>
IOWA CTRS									
I/O <sup>g</sup>	8.0	2.3	5.3	2.3	8.7	2.6	0.6	1.2	<i>a</i> , <i>c</i> > <i>o</i> > <i>n</i>
O/D <sup>h</sup>	4.4	3.1	7.3	2.0	8.1	3.4	0.1	0.3	<i>o</i> , <i>c</i> > <i>a</i> > <i>n</i>

*Note.* AD/HD = Attention Deficit / Hyperactivity Disorder; ODD/CD = Oppositional Defiant Disorder / Conduct Disorder; NC = normal controls; *a* = AD/HD; *o* = ODD/CD; *c* = comorbid; *n* = normal controls; CBCL = Child Behavior Checklist; TRF = Teacher Rating Form; DBD = Disruptive Behavior Disorder Rating Scale; CD = conduct disorder; IOWA CTRS = Iowa Conners Teacher Rating Scale.

<sup>a</sup>Number of males. <sup>b</sup>Student Newman Keuls ( $\alpha$  set at .05). <sup>c</sup>Aggressive Behavior scale. <sup>d</sup>Delinquent Behavior scale. <sup>e</sup>*n* = 23. <sup>f</sup>Impulsivity/Hyperactivity scale. <sup>g</sup>Inattention/Overactivity scale. <sup>h</sup>Oppositional/Defiant scale.

stimulus and stop signal was 250 ms. If the subject inhibited his/her response, the delay on the next stop trial was increased by 50 ms. If the subject failed to inhibit his/her response, the delay on the next stop trial was decreased by 50 ms. By using this tracking algorithm, it was established that all subjects inhibited on an average of 50% of the stop trials. Therefore, on an average the go process and the stop process finish at the same time. Thus, the finishing time of the go process can be used to estimate the SSRT. SSRT can be calculated by subtracting the mean delay from the mean go reaction time.

In addition to SSRT, a number of variables reflecting the response execution process were obtained. These

variables are mean reaction time on go trials (MRT), standard deviation of the reaction times on go trials (*SD*), and the percentage correct responses on go trials. MRT and *SD* were calculated across correct responses on go trials.

### WISC-R

In addition to the stop paradigm, two subtests of the Revised Wechsler Intelligence Scale for Children (WISC-R) were administered to assess intelligence. These subtests were Vocabulary and Block Design. The estimation of the IQ as obtained by these subtests correlates  $r = .90$  with the full scale IQ (Groth-Marnat, 1997).



## Procedure

When subjects entered the experimental room, they were first informed of the purpose of the experiment and of the nature and the duration of the tasks that they were going to perform. Following practice, six experimental blocks were administered, of which three blocks were administered in a reward condition and three in a nonreward condition. The order of reward and nonreward conditions was counter-balanced across groups. Standardized instructions were used. Children were directed to respond as quickly and as accurately as possible, and to inhibit their response when they heard the stop signal. In order to reach an optimal level of task performance, children received feedback on the speed and accuracy of their performance during the practice blocks. Additional instructions for the reward condition were used. Children were informed that they would earn 100 points each time they successfully inhibited. They were informed that the points they earned could be exchanged for a prize. The prizes to be won were shown to the children before performing the stop paradigm in the reward condition. The more points the child earned, the larger the prizes. During task performance, successful inhibition resulted in a 100-point gain, and the experimenter saying "good!" It was ensured that each child earned the same number of points at the end of the reward condition. The tracking mechanism assured that each child would earn about 1200 points in the reward condition. Following the last block of trials, the child was told that (s)he received some extra points for working fast and not waiting for the stop signal, and the experimenter rounded the number of points off to 1500. In the nonreward condition, no points could be earned. A short break was scheduled between the two conditions.

In both conditions, children were required to complete a visual analog scale (Oosterlaan & Sergeant, 1998a). This rating scale was administered following the first block of each condition. The scale was a 100-mm line on which children indicated how motivated they felt in performing the next two blocks of the stop paradigm. The left end of the scale was marked with a sad face, whereas the right end was anchored with a smiling face. The experimenter explained to the children that the sad face meant "not at all motivated," and that the smiling face meant "very much motivated." Children could indicate their level of motivation at any point on the line. Scores on the scale may have a range from 0 (not at all motivated) to 100 (very much motivated).

## Statistical Analyses

The subjective rating scale data (motivation to complete the task) were analyzed using nonparametric tests, because the distribution of data was skewed. To analyze the effect of reward on the subjective level of motivation to complete the task, a Friedman test for

related samples was used. A Kruskal–Wallis test was used to investigate possible group differences.

Measures derived from the stop paradigm (SSRT, MRT, *SD*, and percentage correct responses) were analyzed using ANOVAs with group as the between-subjects factor (4 levels) and condition as a within-subjects repeated factor (2 levels). To interpret the main effects of group, and group by condition interactions, contrast tests were used to compare each psychopathological group with the NC group.

The groups described above differed for sex and IQ. Although these variables did not significantly correlate with the dependent variables, the same ANOVAs were conducted controlling for these variables to check whether the results remained the same.

Since there is debate on which subgroups should be placed within the diagnostic category of AD/HD (Barkley, 1997; Clarke, Barry, McCarthy, & Selikowitz, 1998; Milich, Balentine, & Lynam, 2001), a subgroup analysis was performed. The dependent stop task variables were analyzed using ANOVAs with AD/HD subgroup as the between-subject factor (3 levels) and condition as a within subject factor (2 levels).

AD/HD and ODD/CD are usually considered as categories, but they can also be treated as dimensions. In the categorical approach, cut-off scores for certain disorders are set. However, in the pure AD/HD group subclinical ODD/CD behavior may be present and in the pure ODD/CD group subclinical AD/HD behavior may be present. We felt that a regression analysis would enable interpretation of results to be free of current categorical thresholds. Therefore, in addition to the categorical approach, we used the rating scale data as dimensions and applied a multiple regression analysis to predict the dependent variables using composite measures of AD/HD as well as of ODD/CD. The composite measures were comprised of scale scores on the DBD.

## RESULTS

### Nonparametric Tests for Motivation to Complete the Task

The task manipulation was successful: All subjects felt more motivated to perform the stop paradigm in the reward condition ( $M = 86.0$ ,  $SD = 22.2$ ) than in the nonreward condition ( $M = 67.6$ ,  $SD = 31.3$ ;  $\chi^2(1, N = 111) = 25.8$ ,  $p < .001$ ). Neither in the nonreward condition ( $\chi^2(3, N = 113) = 1.5$ ,  $ns$ ), nor in the reward condition ( $\chi^2(3, N = 113) = 5.0$ ,  $ns$ ) were any group differences detected for the motivation to complete the task (see Table 2).

Table 2. Group Means and Standard Deviations for Stop Paradigm Measures in the Reward and the Nonreward Condition.

Measure	Group							
	AD/HD		ODD/CD		Comorbid <sup>a</sup>		Normal control	
	<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>
Mean reaction time								
Nonreward	417.4	85.0	416.4	71.6	427.5	125.2	362.3	47.3
Reward	457.6	94.3	443.0	79.0	484.4	139.6	380.0	47.8
Variability of reaction times								
Nonreward	91.3	29.4	78.8	31.5	88.1	42.5	58.9	22.8
Reward	95.0	33.8	81.4	25.7	92.4	35.2	59.5	19.3
Percentage correct on go trials								
Nonreward	94.6	5.4	96.8	4.3	94.9	3.9	95.7	5.0
Reward	96.8	7.6	98.4	1.9	98.3	2.2	97.0	3.5
Percentage inhibition								
Nonreward	48.8	5.6	49.8	5.8	50.5	4.5	47.9	4.2
Reward	53.5	4.5	53.4	4.5	54.9	7.2	50.7	3.3
Stop signal reaction time (subtraction) <sup>b</sup>								
Nonreward	173.1	79.0	168.2	60.9	171.2	60.5	153.5	38.9
Reward	157.8	52.9	146.2	34.6	149.4	51.4	147.0	30.5
Stop signal reaction time (integration) <sup>b</sup>								
Nonreward	163.8	92.7	161.2	68.1	161.9	65.0	153.5	41.8
Reward	135.8	58.8	130.7	38.9	126.2	40.0	140.6	35.3
Subjective motivation								
Nonreward	67.4	28.7	73.0	35.2	62.9	35.9	67.7	28.2
Reward	87.5	4.3	88.5	17.3	86.5	27.8	83.5	19.8

Note. AD/HD = Attention Deficit / Hyperactivity Disorder; ODD/CD = Oppositional Defiant Disorder / Conduct Disorder; <sup>a</sup>comorbid = AD/HD + ODD/CD; <sup>b</sup>SSRT was calculated in two ways, accommodating for slight deviations in percentage inhibition, which was aimed at 50% (see text for further details).

## ANOVA

### Response Inhibition

The tracking mechanism was successful: The mean percentage of inhibition was close to the expected 50% rate, that is, 50.6% across groups and conditions. For percentage inhibition, a main effect of condition was detected ( $F(1, 109) = 46.0, p < .001$ ). All groups showed a somewhat higher percentage inhibition in the reward condition (see Table 2). Group differences were noted for percentage inhibition ( $F(3, 109) = 4.4, p < .05$ ). Contrast tests revealed that the group effect was due to the ODD/CD – NC comparison ( $F(1, 109) = 4.7, p < .05$ ), and the comorbid – NC comparison ( $F(1, 109) = 12.2, p = .001$ ). Both the

ODD/CD and the comorbid group had a slightly higher percentage of inhibition than the NC group. This result was not predicted, since the tracking algorithm should ensure that each child in each condition reaches approximately 50% inhibition on the stop trials.

In order to estimate SSRT using the subtraction method suggested by Logan et al. (1997), the percentage of inhibition has to be 50% for each individual. If the percentage of inhibition deviates from 50%, it cannot be assumed that the go process and the stop process finish, on average, at the same time. Since the percentage of inhibition was slightly different for the groups and the two conditions, SSRT was also calculated using the so-called integration method (Logan, 1994).

This method calculates SSRT taking into account individual differences in the percentage of inhibition. For a description of the integration method, see Logan (1994).

A main effect of condition was found for SSRT as calculated by the subtraction method ( $F(1, 109) = 10.6, p < .05$ ). All groups had a faster stop process in the reward condition compared to the nonreward condition (see Fig. 1). Contrary to predictions, no significant group difference was found for SSRT ( $F(3, 109) = 0.7, ns$ ), nor was a significant interaction observed for SSRT between group and condition ( $F(3, 109) = 0.7, ns$ ). This means that all the groups showed a comparable decrease in the latency of their stop process in the reward condition (see Fig. 1). When a contrast test was used to compare the AD/HD group with the NC group, the difference for SSRT did not reach statistical significance ( $F(1, 109) = 3.3, p = .18$ , effect size  $d = .33$ ).

The same analysis was applied to SSRT as calculated by the integration method. This analysis yielded similar results: a main effect of condition ( $F(1, 109) = 23.6, p < .001$ ), no group differences ( $F(3, 109) = 0.1, ns$ ), and no group by condition interaction ( $F(3, 109) = 1.0, ns$ ).

*Response Execution*

The results for measures of response execution are presented in Table 2 and depicted in Figure 1.

A main effect of condition was observed for MRT ( $F(1, 109) = 58.7, p < .001$ ), and for the percentage correct responses on go trials ( $F(1, 109) = 21.3, p < .001$ ). When subjects were rewarded for successful inhibition, their reaction times to go stimuli were slower and they performed their responses with a higher level of accuracy. These findings may be interpreted as a tendency to wait for the stop signal in order to increase the chance to be rewarded in all groups. The reward condition did not have an effect on the variability of reaction times ( $F(1, 109) = 1.3, ns$ ).

A main effect of group was found for MRT ( $F(3, 109) = 6.5, p < .001$ ), and variability of reaction times ( $F(3, 109) = 10.9, p < .001$ ). Contrast tests revealed that the AD/HD group had slower ( $F(1, 109) = 9.3, p < .05$ ), and more variable reaction times ( $F(1, 109) = 23.7, p = .000$ ) than controls. Similarly, the ODD/CD group showed slower ( $F(1, 109) = 6.6, p = .01$ ) and more variable reaction times ( $F(1, 109) = 8.2, p = .005$ ) than controls. Furthermore, children with comorbid AD/HD + ODD/CD exhibited slower reaction times ( $F(1, 109) = 16.3, p < .001$ ) and greater variability in reaction times than control children ( $F(1, 109) = 21.4, p < .001$ ). No significant group differences were detected for accuracy ( $F(3, 109) = 0.99, ns$ ).

A group by condition interaction was found for the speed of the response execution process

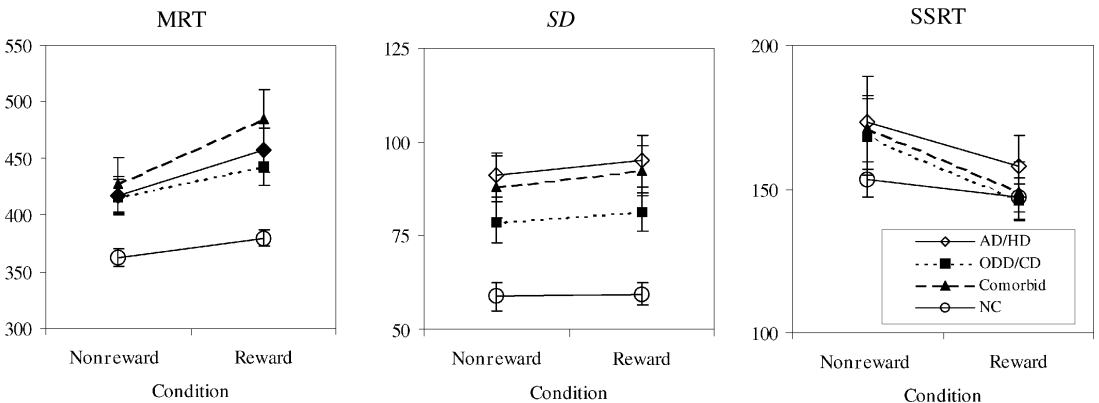


Fig. 1. The stop paradigm variables as a function of condition for Attention Deficit/Hyperactivity Disorder (AD/HD), Oppositional Defiant Disorder/Conduct Disorder (ODD/CD), for comorbid AD/HD + ODD/CD, and for normal controls (NC). Mean reaction time (MRT) is depicted in the left panel, standard deviation of reaction times (SD) is depicted in the middle panel, and stop signal reaction time (SSRT) is shown in the right panel.

( $F(3, 109) = 4.0, p = .01$ ). Contrast tests indicated that the interaction effect for MRT was due to the AD/HD + ODD/CD – NC comparison ( $F(1, 109) = 11.0, p = .001$ ). As depicted in Figure 1, the comorbid group slowed down more in the reward condition as compared to controls. The interaction effect for the AD/HD – NC comparison did not reach statistical significance, but a tendency was observed ( $F(1, 109) = 3.4, p = .07$ ) for children with AD/HD to slow down in the reward condition to a greater extent than control children. Children with ODD/CD did not slow down more than control children in the reward condition ( $F(1, 109) = .5, p = .5$ ). These findings may be interpreted as a stronger tendency to seek reward in both the AD/HD and AD/HD + ODD/CD groups compared to the NC group.

### Analysis Controlling for IQ and Gender

The group difference for sex was controlled for by excluding all but 5 girls (selected randomly) from the control group. IQ was controlled for by excluding all children with an IQ lower than 80. Since group differences for IQ were still observed, IQ was entered as a covariate in the analysis. Main effects and interaction effects for all dependent variables remained the same.

### AD/HD Subgroup Analyses

No differences emerged between AD/HD subgroups for SSRT ( $F(2, 19) = .74, ns$ ), percentage inhibition ( $F(2, 19) = .37, ns$ ), MRT ( $F(2, 19) = 0.76; ns$ ), variability of reaction times ( $F(2, 19) = 1.7, ns$ ), and percentage correct ( $F(2, 19) = .29, ns$ ). Importantly, AD/HD subgroups did not differentially react to reward: None of the subgroup by reward interactions were significant.

### Multiple Regression Analyses

In this section, AD/HD and ODD/CD symptoms are considered from a dimensional rather than a categorical approach (Nigg, Hinshaw, Carte, & Treuting, 1998). It was expected that regression models would provide converging evidence with the previously described results using ANOVAs.

Two stepwise univariate regression models were run for each condition to investigate the relative contribution of AD/HD and ODD/CD ratings to the proportion explained variance of

SSRT and the measures for response execution. To control for a possible confounding effect of age on the predictors AD/HD and ODD/CD, age was entered at step 1. Since AD/HD and ODD/CD symptoms were highly correlated ( $r = .76$ ), two regression models were run. In the first model, a composite measure of AD/HD was entered at step 2, and a composite measure of ODD and CD at step 3. The ODD/CD predictor could not account for much variance in the dependent variable because it was entered as the last step. In the second model, ODD/CD symptoms were entered at step 2, and AD/HD symptoms were entered at step 3. The composite AD/HD score was created by calculating the mean of the parent DBD Inattention and Impulsivity/Hyperactivity scales, and the teacher DBD Inattention and Impulsivity/Hyperactivity scales. The composite ODD/CD score was created by calculating the mean of the parent DBD ODD and CD scales and the teacher DBD ODD and CD scales.

Contrary to the predictions, no relevant proportion of the variance in SSRT was accounted for by AD/HD symptoms in either of the conditions (see Table 3). The proportion of variance in MRT explained by AD/HD symptoms (step 2) was 18% ( $p < .001$ ) in the nonreward condition, and 24% in the reward condition ( $p < .001$ ). AD/HD symptoms explained 25% variance in the nonreward condition ( $p < .001$ ), and 26% in the reward condition ( $p < .001$ ) for variability of reaction times. No relevant proportion of the variance for accuracy was accounted for by AD/HD. Furthermore, ODD/CD symptoms entered at step 3 did not account for any additional proportion of variance for any of the variables.

Thus, the regression analyses showed that AD/HD symptoms have power in predicting response execution measures but not in predicting response inhibition. This finding is in agreement with the results of the ANOVAs reported above: The AD/HD group showed slower reaction times with greater variability compared to the NC group, but similar SSRTs. In the reward condition, the proportion of variance in MRT that is accounted for by AD/HD increases compared to the nonreward condition. This finding is in agreement with the results obtained with ANOVAs: the AD/HD group and the comorbid AD/HD + ODD/CD

group (but not the ODD/CD group) slowed down more in the reward condition than in the nonreward condition compared to controls. This suggests that the interactions found were due to the AD/HD symptoms.

When the order of entry of the predictors AD/HD and ODD/CD was reversed, ODD/CD symptoms could not explain a relevant proportion of variance in SSRT nor in accuracy. Furthermore, ODD/CD symptoms accounted for relevant proportions of variance in MRT and variability of reaction times. This finding is in agreement with the results of the ANOVAs: the ODD/CD and the AD/HD + ODD/CD groups showed slower reaction times with greater variability than the control group. However, the proportion of variance in MRT and *SD* accounted for by ODD/CD was smaller than the proportion of variance in MRT and *SD* accounted for by AD/HD in the first model (see Table 3). It was found that the power of ODD/CD in predicting MRT and *SD* did not increase in the reward condition in comparison with the nonreward condition. The proportion of variance in MRT explained by ODD/CD symp-

toms (step 2) was 13% in the nonreward condition ( $p < .001$ ), and 16% in the reward condition ( $p < .001$ ). ODD/CD symptoms accounted for 15% of variance for variability of reaction times in the nonreward condition ( $p < .001$ ), and for 16% in the reward condition ( $p < .001$ ). In this model it was found that AD/HD symptoms (entered at step 3) could explain additional variance in MRT and *SD*. For MRT, AD/HD explained 5% additional variance in the nonreward condition ( $p < .05$ ), and 8% in the reward condition ( $p < .001$ ). AD/HD accounted for 10% additional variance for variability of reaction times in the nonreward condition ( $p < .001$ ) and 10% in the reward condition ( $p < .001$ ).

These findings suggest that symptoms of AD/HD and ODD/CD are not powerful predictors of the latency of the inhibitory process. It is suggested that AD/HD is a more powerful predictor for measures of response execution than ODD/CD, and that AD/HD explains variance in MRT and variability of reaction times, after having controlled for the predictive power of ODD/CD behavior. However, the predictive power of ODD/

Table 3. Multiple Regression Analyses With the Predictors Age Entered at Step 1, AD/HD Entered at Step 2, and ODD/CD Entered at Step 3 (Model 1), and ODD/CD Entered at Step 2, and AD/HD Entered at Step 3 (Model 2).

Predictor	Dependent measures											
	MRT			<i>SD</i>			SSRT			Percentage correct on go trials		
	$\beta$	R <sup>2</sup>	$\Delta R^2$	$\beta$	R <sup>2</sup>	$\Delta R^2$	$\beta$	R <sup>2</sup>	$\Delta R^2$	$\beta$	R <sup>2</sup>	$\Delta R^2$
Nonreward												
Step 1, age	-.30	.06	.06*	-.24	.03	.03*	-.12	.01	.01	.19	.04	.04*
Step 2, AD/HD	.36	.24	.18**	.49	.28	.25**	.08	.02	.01	-.17	.04	.005
Step 3, ODD/CD	.09	.24	.00	.02	.28	.00	.01	.02	.00	.13	.05	.01
Step 2, ODD/CD	.09	.19	.13**	.02	.18	.15**	.01	.02	.01	.13	.04	.00
Step 3, AD/HD	.36	.24	.05*	.49	.28	.10**	.08	.02	.00	-.17	.05	.01
Reward												
Step 1, age	-.20	.02	.02	-.25	.04	.04*	-.35	.11	.11**	.23	.06	.06*
Step 2, AD/HD	.42	.26	.24**	.49	.30	.26**	.07	.12	.01	-.02	.06	.00
Step 3, ODD/CD	.09	.26	.00	.03	.30	.00	.03	.12	.00	.09	.07	.00
Step 2, ODD/CD	.09	.18	.16**	.03	.20	.16**	.03	.12	.01	.09	.07	.01
Step 3, AD/HD	.42	.26	.08**	.49	.30	.10**	.07	.12	.00	-.02	.07	.00

Note. AD/HD = Attention Deficit / Hyperactivity Disorder; ODD/CD = Oppositional Defiant Disorder / Conduct Disorder; MRT = mean reaction time; *SD* = variability of reaction times; SSRT = stop signal reaction time.

\* $p < .05$ .  
\*\* $p < .001$ .

CD behavior for measures of response execution seems to be dependent on the correlation with AD/HD behavior. AD/HD symptoms have more power in predicting MRT in the reward condition than in the nonreward condition. In contrast, the predictive power of ODD/CD does not increase in the reward condition in comparison with the nonreward condition.

## DISCUSSION

The four main findings of this study were the following: (a) our attempt to enhance children's motivation to complete the task in the reward condition was successful, (b) none of the DBD groups showed a deficit in response inhibition, (c) the comorbid AD/HD+ODD/CD group and the AD/HD group slowed down more dramatically than controls in the reward condition as compared to the nonreward condition, and (d) the findings for the three AD/HD subgroups were the same as the findings for the AD/HD group as a whole.

All the participants performed the task more efficiently in the reward condition than in the nonreward condition. This suggests that the reward manipulation was successful. In addition, all children inhibited faster, showed a slightly higher percentage of inhibition, reacted more slowly, and made less errors in the reward condition as compared to the nonreward condition. Thus, overall performance was better in the reward condition than in the nonreward condition, except for the speed of the response execution process, which was slower. This finding may be explained by the fact that reward was given after successful inhibition, and not after fast response execution. This may have induced a response bias towards inhibition, at the cost of responding fast. Children slowed down their responses (waited for the stop signal), in order to increase the chance to inhibit (and the chance to be rewarded). It was shown that children indeed inhibited on slightly more stop trials in the reward condition than in the nonreward condition. The latter finding was not expected, since the tracking mechanism should insure that the percentage of inhibition equals 50% for each child in each condition. This result suggests that the tracking

mechanism could not fully catch up with the children's strategy.

In the current study, previous findings of slow response inhibition in AD/HD were not replicated (e.g., Nigg, 1999; Oosterlaan et al., 1998; Schachar et al., 2000). Although the AD/HD group showed slower SSRTs than normal controls, this difference was not significant. The nonsignificant difference between the AD/HD and the NC group translated into a small effect size ( $d = 0.33$ ). Although the inhibition deficit in AD/HD children found in previous studies seems to be a robust finding with a medium effect size ( $d = 0.64$ ; Oosterlaan et al., 1998), this is not the first study that fails to find a difference between AD/HD children and controls on SSRT (Daugherty, Quay, & Ramos, 1993; Kuntsi, Oosterlaan, & Stevenson, 2001; Pliszka, Liotti & Woldorff, 2000). Thus, the present finding, although consistent with findings of some other studies, requires consideration of how the current paradigm differs from the majority of previous reports using the stop paradigm.

A factor that is possibly responsible for this failure to replicate, is the type of stop paradigm used here. In the current study, a stop paradigm with a tracking algorithm was used, which dynamically varied the delay between go and stop signal, contingent on the child's inhibitory performance. This results in an inhibition rate of approximately .5 in all children. In most previous studies that reported group differences on the speed of the inhibitory process, a version of the stop paradigm was used with a number of fixed delays (usually four) between the presentation of the go stimulus and the stop signal (but see Chhabildas et al., 2001; Nigg, 1999; Schachar et al., 2000; Scheres et al., 2001). This results in four different inhibition rates and these rates can be different between subjects. In this study, the stop paradigm with tracking algorithm was employed, since it has been demonstrated that this procedure has several methodological and practical advantages compared to the fixed delay procedure (Band, 1997). However, it is possible that the stop paradigm with the tracking algorithm in one way or another does not measure the same SSRT as the paradigm with fixed delays. It is noted here that the SSRT as obtained by the

current task and procedure is in fact more reliable than the SSRT as measured in previous stop paradigm research (Band, 1997). In a previous study employing the stop paradigm with a tracking mechanism, it was demonstrated that children with relatively high levels of externalizing behavior had impaired inhibitory control (Kooijmans, Scheres, & Oosterlaan, 2000). In four other studies on response inhibition in AD/HD, the stop paradigm with tracking mechanism was used (Chhabildas et al., 2001; Nigg, 1999; Schachar et al., 2000; Scheres et al., 2001). Group differences between children with AD/HD and a normal control group on SSRT were demonstrated in three out of these four studies. The latter findings would argue for convergence between the two paradigms.

A second possible explanation for our failure to replicate poor response inhibition in AD/HD, is that there was not enough power to detect group differences. In a meta-analysis, Oosterlaan et al. (1998) reported a medium effect size for SSRT differences between AD/HD and normal controls ( $d = 0.64$ ). To detect this effect with a power of 0.80, 22 subjects are required for each group. This requirement was met here and thus the groups were sufficiently large to measure the expected difference between AD/HD and normal controls for SSRT.

An alternative argument to explain our findings could be that the pathological groups were not severely impaired. This argument, however, seems unlikely for four reasons. First, the groups were clearly different on the relevant parent and teacher rating scales. Second, the inclusion criterion of pervasiveness was applied to all pathological groups. Third, samples were drawn from children who attended special school services for children with extreme behavioral problems (2.2% of Dutch children in the age range of 6–12 years attend these school services). Fourth, the DBD groups differed from the normal controls on the go process. All in all, it seems unlikely that the pathological groups were not significantly impaired.

It has been hypothesized that a deficit in response inhibition is only observed in children with AD/HD combined subtype or AD/HD hyperactive/impulsive subtype (Barkley, 1997). It could

therefore be argued that a deficit in response inhibition in AD/HD was not observed in the current study, since the AD/HD group did not consist of only children with AD/HD combined subtype, but included children with AD/HD inattentive subtype. However, when only the children with AD/HD combined subtype (with and without comorbid ODD/CD) were compared with normal controls on SSRT, no group differences emerged, and the effect size was small. This finding remained the same when symptoms of ODD/CD were controlled for (data available from the first author).

Finally, since the DBD was used as a selection instrument in the current study, it is not known whether possible comorbid internalizing problems may have influenced the present results. Recently, it was shown that children with relatively high levels of internalizing problems showed enhanced response inhibition (Kooijmans et al., 2000). Oosterlaan and Sergeant (1998b) found some suggestion for enhanced response inhibition in anxious children, although this effect was not significant. Therefore, it could be argued that in the current study, possible comorbid internalizing problems may have played a role in obtaining normal SSRTs in the AD/HD group. Future research should take into account comorbid internalizing problems.

The stop paradigm is purported to measure prepotent response inhibition. Several other forms of inhibition have been distinguished. Barkley (1997) distinguished between prepotent response inhibition, ongoing response inhibition, and interference control. Nigg (2000) suggested interference control, cognitive inhibition, behavioral (or prepotent) inhibition, and oculomotor inhibition to be four forms of executive inhibition. Sergeant et al. (1999) reviewed 12 paradigms measuring response inhibition. They concluded that for five operationalizations there was no evidence for a response inhibition deficit in AD/HD. For four operationalizations some support was found for a deficit in response inhibition in AD/HD. With only three paradigms unequivocal evidence favoring the hypothesis of a response inhibition deficit in AD/HD was obtained. Against that background, group differences between AD/HD and normal control children on the latency of the inhibition

process as measured with the stop paradigm should be placed into the category of "some support" for the inhibition deficit in AD/HD.

The second aim of the current study was to explore whether poor response inhibition is a core problem in children with AD/HD, or, alternatively, whether it is a manifestation of an underlying reward deficit. Because children with AD/HD did not show a deficit in response inhibition, this question could not be addressed directly. However, a group by condition interaction was found for MRT: It was shown that children with comorbid AD/HD+ODD/CD and children with AD/HD slowed down more in the reward condition than in the nonreward condition compared to controls. In line with this finding, the regression analysis showed that the power of AD/HD symptoms in predicting response times increased in the reward condition compared to the nonreward condition, whereas this was not the case for ODD/CD symptoms. This finding could be interpreted as a strategy effect: children with AD/HD (with or without comorbid ODD/CD) seem to be more willing to ignore instructions, when they are rewarded for successful inhibition, and, therefore, to slow down in order to improve their inhibitory performance and obtain more reward. In terms of the specificity of the role of reward, this finding suggests that a stronger tendency to seek reward is specifically related to AD/HD symptoms. The finding that attempting to obtain immediate reward is related to AD/HD symptoms is in line with previous findings (e.g., Carlson, Mann, & Alexander, 2000; Douglas & Parry, 1994; Sonuga-Barke, Taylor, Sembi, & Smith, 1992; Tripp & Alsop, 1999). This stronger tendency to seek immediate reward in AD/HD might reflect a role of the reward circuitry in AD/HD: dopamine neurons of the VTA and substantia nigra, which are connected to brain structures involved in motivation such as the striatum, nucleus accumbens, and frontal cortex (Schultz et al., 1997).

In a recent study on the effects of contingencies on response inhibition in AD/HD, an inhibition deficit in AD/HD was reported (Slusarek, Velling, Bunk, & Eggers, 2001). In that study, it was shown that slow SSRTs in AD/HD were only observed in a condition with low incentives. Inhibitory performance in the AD/HD group nor-

malized in a condition with high incentives. Slusarek et al.'s study and the current study differ on a number of aspects: Firstly, in Slusarek et al.'s study, feedback was given after *every* trial (also after go trials). Secondly, Slusarek et al. included a low-and a high-incentive condition, rather than a reward and nonreward condition. Slusarek et al. studied the effect of motivational *level* within children. Thirdly, in the low-incentive condition, children lost 1 point when they failed to inhibit, and in the high-incentive condition, children lost 5 points when they failed to inhibit. This manipulation suggests that in Slusarek et al.'s study the effect of response *cost* were investigated rather than the effect of *reward*.

The lack of reward dominance in the ODD/CD group is not in line with the few studies that have shown children with ODD or CD to be more reward dominant than control children (O'Brien & Frick, 1996; O'Brien, Frick, & Lyman, 1994; Shapiro, Quay, Hogan, & Schwartz, 1988). A possible explanation is that in the current study another paradigm was used to measure the effect of reward on task performance. A study by Oosterlaan and Sergeant (1998a) employing the stop paradigm failed to show reward dominance in children with aggressive behavioral disorders and also in children with AD/HD. Only a few studies addressed the issue of the specificity of the effect of reward on children with AD/HD, ODD, and CD (O'Brien & Frick, 1996; Oosterlaan & Sergeant, 1998a). Two other studies included a DBD group consisting of children with comorbidity for AD/HD, ODD, and/or CD (Carlson & Tamm, 2000; O'Brien et al., 1994). To clarify the specificity of reward dominance in children with DBD, we suggest that future research should include groups of children with pure AD/HD, ODD/CD and comorbid DBD.

It was shown that the three subgroups of children with AD/HD performed equally on the stop paradigm. In addition, the effect of reward was the same for the three AD/HD subgroups. These findings do not support the notion that AD/HD combined subtype and AD/HD inattentive subtype are distinct and unrelated disorders (e.g., Barkley, Grodzinsky, & DuPaul, 1992; Milich et al., 2001). However, these findings should be interpreted with caution and definitive



conclusions should not be drawn, given the small number of children in the AD/HD subgroups. Since there is some evidence that AD/HD inattentive subtype is a valid AD/HD subtype (e.g., Carlson, Shin, & Booth, 1999), and since there is debate on this issue, research that compares the three subtypes on key measures such as response inhibition is clearly important and required.

The main features by which the clinical and the normal control groups could be distinguished were response execution measures: The three clinical groups demonstrated slower reaction times with greater variability in responding. This robust finding has been interpreted previously as evidence for a problem in the output stages of information processing in AD/HD, which might be related to inadequate resource allocation (Sergeant et al., 1999). Although thus far there has been little evidence for a deficit in early information processing stages in AD/HD (Sergeant & Van der Meere, 1990b), recent findings of an ERP study on inhibition in AD/HD (Brandeis et al., 1998) suggested that children with AD/HD show altered initial orienting to the go stimulus in the stop paradigm. This altered early orienting negativity was associated with failures to inhibit in AD/HD. Given these data, an alternative way of interpreting the slow reaction times in the clinical groups would be that children with DBD demonstrate slower reaction times, because they have an altered orienting to the go stimulus. Future research has to further clarify the possible relation between early information processing stages (such as orienting) and inhibitory control.

In sum, contrary to the predictions, AD/HD children did not show a slow response inhibition process, neither did children with ODD/CD or comorbid AD/HD + ODD/CD. More research has to show whether this lack of replication is due to the version of the stop paradigm (with tracking mechanism) that was used. If this finding is replicated, it will have considerable implications for the generality of the inhibition hypothesis in AD/HD. Reward did not differentially affect the groups on the following variables: speed of the inhibitory process (SSRT), percentage inhibition, variability of reaction times, and accuracy. However, it did affect groups differentially on the speed of the response execution process (MRT).

This finding may be interpreted as a tendency to seek reward in children with AD/HD with or without comorbid ODD/CD. This tendency seems to be specifically related to symptoms of AD/HD, since it was not observed in the pure ODD/CD group. When symptoms of DBD were treated as dimensions as opposed to categories, our conclusions remained the same. There was no difference in performance between three subgroups of AD/HD.

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